15-deoxy-$\Delta^{12,14}$-prostaglandin J_2 inhibits cell migration of renal cell carcinomas independently of PPARγ and CRTH2

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Renal cell carcinoma (RCC) accounts for 2–3% of all malignant tumors. Even with oncologic removal, about 40% of patients will develop metastases after surgical resection. The five-year survival probability of patients with metastatic renal cell carcinoma is less than 10% because of the cancer's resistance to chemotherapy and radiotherapy. Thus, there is an urgent need to establish novel therapeutic approaches for metastatic RCC treatment. The metastatic cascade has been reported to be modulated by an endogenous carcinostatic 15-deoxy-$\Delta^{12,14}$-prostaglandin J_2 (15d-PGJ_2). A nuclear receptor of 15d-PGJ_2 is peroxisome-proliferator activated receptor γ (PPARγ), and its membrane receptor is chemoattractant receptor-homologous molecule expressed on Th2 cells (CRTH2). 15d-PGJ_2 has also been reported to reduce cell migration, stimulate focal adhesion disaggregation, and induce filamentous actin realignment. In the present study, we evaluated the effects of 15d-PGJ_2 on the migration of Caki-2 RCC cells. Although treatment with low concentrations of 15d-PGJ_2 did not cause apoptosis, it did decrease the migration of Caki-2 cells. PPARγ and CRTH2 did not mediate the inhibitory effect of 15d-PGJ_2 on the migration of Caki-2 cells. Our present study proposes the therapeutic potential of 15d-PGJ_2 for prevention of RCC metastasis.